

A case of primary neuroleptospirosis

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Introduction

The clinical spectrum of leptospirosis ranges from inapparent infection to fulminant and fatal disease. It is uncommon for leptospirosis to present as a primary neurological disease [1]. We describe a patient presenting with transverse myelitis and papilloedema due to leptospirosis.

Case report

A 30-year-old paddy farmer from Thelijjawala, Matara was transferred from Teaching Hospital, Karapitiya to National Hospital of Sri Lanka with sudden onset of paraparesis, urinary retention, and numbness below his upper abdomen of three weeks duration. He did not have symptoms referable to his upper limbs and cranial nerves, seizures or loss of consciousness.

On admission to our unit, he had spastic paraparesis with grade 4 muscle power and extensor plantar responses. A sensory level with impairment of pain, touch, temperature and joint position sense was present at the D8 segmental

level. Sacral sensation was lost. The gait was spastic, but he was able to walk with support. There were no neurological deficits in the upper limbs. Coordination was normal. He had urinary retention, and bilateral papilloedema. Higher cortical functions and other cranial nerves were normal.

Except for a non-tender, firm enlargement of the liver extending 3 cm below the right costal margin, the rest of physical examination was normal.

Magnetic resonance imaging (MRI) scan of the dorsal spine excluded a compressive myelopathy and demyelination. A contrast-enhanced computed tomography (CT) of the brain excluded an intracranial space-occupying lesion.

The cerebrospinal fluid (CSF) was clear with 40 mg/dl of proteins, 34/ μ L of lymphocytes and 4.9 mmol/L of glucose (Blood glucose 6.2 mmol/L). A polymerase chain reaction test for *Mycobacterium tuberculosis* was negative. Blood counts and blood picture showed neutrophil leucocytosis.

Ultrasound scan of the abdomen showed a 3 cm hepatomegaly of uniform echo-pattern and no focal

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lesions. Hepatic transaminases were elevated—AST: 147 iu/L (normal 5–40 iu/L) and ALT: 347 iu/L (normal 5–40 iu/L). The other liver function tests were normal. The elevated transaminases normalised in the subsequent two weeks. ESR was 6 mm/h. The mantoux test was negative. Urinalysis, serum urea, electrolytes, creatinine and fasting blood sugar estimations were within normal limits. HIV antibody test, VDRL, mycoplasma antibody titre in serum, serology for Hepatitis A and B viruses and Paul Bunnell tests were negative. Visual evoked potentials were normal.

Titres of leptospira agglutination lysis tests performed on admission, 11 days later and 17 days later were 200, 200 and none respectively, thus confirming a recent leptospira infection. Urine darkground microscopy for leptospira was negative.

He was given a course of intravenous crystalline penicillin and dexamethasone. At the time of discharge, 35 days after admission, he was independently mobile with minimum support and had regained bladder control.

Discussion

Leptospirosis has a worldwide distribution, predominantly involving tropical areas. The clinical spectrum of leptospirosis ranges from a mild, anicteric febrile illness to the more serious Weil syndrome, comprising jaundice, renal dysfunction and bleeding diathesis. Our patient presented with transverse myelitis without clinical features of renal dysfunction.

Leptospire reach the CSF and brain as early as 48 h after inoculation [2]. However, nervous system involvement is essentially immune mediated. Neurological manifestations in leptospirosis include aseptic meningitis, encephalitis, intracranial bleeding (subarachnoid and extradural haemorrhage), cerebellitis, movement disorders, myelitis, flaccid paraplegia, mononeuritis, autonomic lability and polymyositis [2]. However, it is uncommon for leptospirosis to present as a primary neurological disease without

clinical features to suggest leptospirosis (primary neuroleptospirosis).

In a series of 100 hospitalised patients in Manila, Philippines, with aseptic meningoencephalitis, five patients were diagnosed to have leptospirosis [3]. In a series reported from Kerala, India, 40 patients presenting with an acute neurological disease were found to have leptospirosis [1]. None of these patients had renal dysfunction or jaundice at presentation. Papilloedema is a documented ocular manifestation of leptospirosis.

Hence, all the features in our patient were consistent with the diagnosis of primary neuroleptospirosis. Had it not been for the high index of suspicion in view of his occupational hazard of being exposed to stagnant water, and a literature search for an association between leptospirosis and myelitis, the diagnosis of leptospirosis would have been easily overlooked.

The prognosis after primary neuroleptospirosis is generally good, but altered sensorium and seizures herald a worse prognosis [2]. Crystalline penicillin has been shown to reduce severity and duration of illness, even in the immune phase [4]. The role of steroids is controversial.

References

1. Panicker JN, Mammachan R, Jayakumar RV. Primary neuroleptospirosis. *Postgraduate Medical Journal* 2001; **77**: 589–90
 2. Bharucha NE, Bharucha EP, Bhabha SK. Infections of nervous system. In: Bradley WG, Daroff RB, Fenichel GM, Marsden CD, eds. *Neurology in Clinical Practice*. Boston: Butterworth-Heinemann, 1996: 1214–5.
 3. Watt G, Manaloto C, Hayes CG. Central nervous system leptospirosis in the Philippines. *Southeast Asian Journal of Tropical Medicine and Public Health* 1989; **20**: 265–9.
 4. Watt G, Padre LP, Tuazon ML, Calubagnib C, Santiago E, eds. Placebo controlled trial of intravenous penicillin for severe and late leptospirosis. *Lancet* 1988; **1**: 433–5.
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